

Ulster Paediatric Society

Junior Members' Forum

The Junior Members' Forum provides opportunity for paediatricians in training to present scientific and clinical material of research interest. At the Annual Meeting in November 1993 Professor Sir David Hull, President of the British Paediatric Association, and Dr Paul Thomas, President of the Ulster Paediatric Society acted as judges. Prizes were awarded on the basis of the best scientific paper to Mr Bill McCallion for his paper "*Helicobacter Pylori* in children in Northern Ireland", and for the best presentation to Dr Fiona Stewart for her paper "Molecular Investigation of patients with Prader Willi syndrome".

Helicobacter Pylori in Children in Northern Ireland – Initial Impressions.

W A McCallion, N F Stirling, K B Bamford*, S R Potts, V E Boston.

Royal Belfast Hospital For Sick Children, 180 Falls Road, Belfast BT12 6BE.

*Department of Microbiology, The Queen's University Of Belfast.

Helicobacter pylori is a recognised cause of gastritis and peptic ulceration and has been implicated in the causation of gastric carcinoma. The prevalence of *Helicobacter pylori* among school-children in developed countries is 1 to 8%. Whilst peptic ulceration in children is uncommon, *Helicobacter pylori* is believed to be a cause of dyspepsia and possibly recurrent abdominal pain. The aim of this paper is to determine: (1) the prevalence of the infection in children in Northern Ireland, (2) the extent of intrafamilial clustering, (3) if prevalence is associated with social class, (4) if *Helicobacter pylori* causes recurrent abdominal pain, and (5) the effect of eradication on gastrointestinal symptoms.

367 children were investigated for *Helicobacter pylori*: 71 presenting with severe dyspepsia had oesophagogastroduodenoscopy, 50 siblings underwent a carbon-13 urea breath test, and 242 children attending for non-gastrointestinal daycase surgery had serological tests only.

	Patients				Social class						Intrafamilial clustering
	Overall	Dyspepsia	Sibs	Daycases	I	II	IIIN	IIIM	IV	V	
Prevalence %	37	45	58	30	0	29	27	47	44	42	96

Of 32 children with severe dyspepsia and *Helicobacter pylori* 2 had peptic ulcers. Following eradication, dyspepsia was cured in one third, persisted with some improvement in one third, and persisted with no improvement in one third. 20% of children with no *Helicobacter pylori* had recurrent abdominal pain compared with 28% of those with *Helicobacter pylori* (p=0.13).

The prevalence of *Helicobacter pylori* in children in Northern Ireland is significantly greater than in any other published report from the developed world. Prevalence is higher in lower social classes ($p=0.01$). Intrafamilial clustering is very common suggesting person-to-person spread. Most children with severe dyspepsia do not have *Helicobacter pylori*. Furthermore eradication of the organism does not alleviate symptoms in the majority recurrent abdominal pain more common in children with *Helicobacter pylori*. Given that peptic ulceration is uncommon despite a very high prevalence of *Helicobacter pylori*, its role as a significant pathogen in children is questioned.

Molecular investigation of patients with Prader Willi syndrome

F J Stewart, A E Hughes, N C Nevin, Northern Ireland Regional Genetics Service, Department of Medical Genetics, Belfast City Hospital, Belfast BT9 7AB.

A study was undertaken of all patients in Northern Ireland with suspected Prader Willi syndrome from a clinical, cytogenetic, and molecular viewpoint. Where possible patients were examined clinically, and blood taken from the patient and both parents for cytogenetic and molecular analysis. Molecular analysis was carried out using two microsatellites 4-3R and GabaRB3 to look for deletions of the Prader Willi region of chromosome 15, or parental disomy.

Results are available on 12 patients: 8 have the typical Prader Willi syndrome diagnosed clinically, 2 have suspected Prader Willi syndrome; 2 have chromosome rearrangements involving 15q12.

Of the 8 patients, 4 showed a deletion on the paternally derived chromosome 15; 2 showed maternal disomy and 2 were uninformative. Of the 2 suspected patients, 1 showed evidence of non-paternity and the other showed no evidence of deletion or disomy. Of the 2 patients with chromosome rearrangements, 1 showed a deletion of 4-3R. Results on the second patient are rather unusual and further work on this sample is planned.

As only 50% of patients with Prader Willi syndrome show a visible cytogenetic deletion we feel that the use of microsatellites is very helpful in confirming the diagnosis – particularly in the newborn period when the clinical features may not be striking.